

FILE 'HOME' ENTERED AT 12:12:05 ON 11 NOV 2009

=> FIL REGISTRY	SINCE FILE	TOTAL
COST IN U.S. DOLLARS	ENTRY	SESSION
FULL ESTIMATED COST	0.22	0.22

FILE 'REGISTRY' ENTERED AT 12:12:35 ON 11 NOV 2009
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provided by InfoChem.

STRUCTURE FILE UPDATES: 10 NOV 2009 HIGHEST RN 1192107-32-6
DICTIONARY FILE UPDATES: 10 NOV 2009 HIGHEST RN 1192107-32-6

New CAS Information Use Policies, enter HELP USAGETERMS for details.

TSCA INFORMATION NOW CURRENT THROUGH June 26, 2009.

Please note that search-term pricing does apply when
conducting SmartSELECT searches.

REGISTRY includes numerically searchable data for experimental and
predicted properties as well as tags indicating availability of
experimental property data in the original document. For information
on property searching in REGISTRY, refer to:

<http://www.cas.org/support/stngen/stndoc/properties.html>

=> E "ARSENIC"/CN 25
E1 1 ARSENIAN STIBIOLUZONITE/CN
E2 1 ARSENIAN STRONTIAN GAMAGARITE/CN
E3 1 --> ARSENIC/CN
E4 1 ARSENIC (+3 OXIDATION STATE) METHYLTRANSFERASE (ALCANIVORAX BORKUMENSIS STRAIN SK2)/CN
E5 1 ARSENIC (AS1+)/CN
E6 1 ARSENIC (AS2)/CN
E7 1 ARSENIC (AS3)/CN
E8 1 ARSENIC (AS4)/CN
E9 1 ARSENIC (AS41+)/CN
E10 1 ARSENIC (III)/CN
E11 1 ARSENIC (III) METHYLTRANSFERASE (OIKOPLEURA DIOICA CLONE BACOIKO007-10X111)/CN
E12 1 ARSENIC (V) OXIDE/CN
E13 1 ARSENIC 0-0.1, CARBON 0.2, CHROMIUM 0.4-0.6, COPPER 0-0.2, IRON 96-98, MANGANESE 0.6-1, MOLYBDENUM 0.2, NICKEL 0.4-0.8, SILICON 0.2-0.4, VANADIUM 0-0.1/CN
E14 1 ARSENIC 0-0.1, COPPER 99.9-100 (ATOMIC)/CN
E15 1 ARSENIC 0-0.2, CADMIUM 100/CN
E16 1 ARSENIC 0-1, CARBON 7, IRON 79-80, PHOSPHORUS 13 (ATOMIC)/CN
E17 1 ARSENIC 0-1, GERMANIUM 45-55, TELLURIUM 45-55 (ATOMIC)/CN
E18 1 ARSENIC 0-1, NICKEL 99-100 (ATOMIC)/CN
E19 1 ARSENIC 0-1.5, SILVER 98.5-100 (ATOMIC)/CN
E20 1 ARSENIC 0-2, LEAD 98-100/CN
E21 1 ARSENIC 0-2, COPPER 90.5-92.5, INDIUM 7.5 (ATOMIC)/CN
E22 1 ARSENIC 0-21, TIN 79-100 (ATOMIC)/CN
E23 1 ARSENIC 0-22, LEAD 78-100 (ATOMIC)/CN
E24 1 ARSENIC 0-25, BORON 0-25, PALLADIUM 75 (ATOMIC)/CN

E25 1 ARSENIC 0-3.1, SILVER 96.9-100 (ATOMIC)/CN

=> S E3

L1 1 ARSENIC/CN

=> DIS L1 1 IDE

THE ESTIMATED COST FOR THIS REQUEST IS 2.05 U.S. DOLLARS
DO YOU WANT TO CONTINUE WITH THIS REQUEST? (Y)/N:Y

L1 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2009 ACS on STN

RN 7440-38-2 REGISTRY

ED Entered STN: 16 Nov 1984

CN Arsenic (CA INDEX NAME)

OTHER NAMES:

CN Arsenic black

CN Arsenic-75

DR 55624-62-9, 39277-51-5

MF As

CI COM

LC STN Files: ADISNEWS, AGRICOLLA, ANABSTR, AQUIRE, BIOSIS, BIOTECHNO, CA,

CABA, CAPLUS, CASREACT, CBNB, CHEMCATS, CHEMLIST, CIN, CSCHEM, CSNB,

DDFU, DETHERM*, DRUGU, EMBASE, ENCOMPLIT, ENCOMPLIT2, ENCOMPPAT,

ENCOMPAT2, HSDB*, IFICDB, IFIPAT, IFIUIDE, IPA, MEDLINE, MRCK*,

MSDS-OHS, PIRA, PROMT, PS, RTECS*, TOXCENTER, TULSA, ULIDAT, USPAT2,

USPATFULL, VETU

(*File contains numerically searchable property data)

Other Sources: DSL**, EINECS**, TSCA**

(**Enter CHEMLIST File for up-to-date regulatory information)

As

PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

99468 REFERENCES IN FILE CA (1907 TO DATE)

3642 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA

99738 REFERENCES IN FILE CAPLUS (1907 TO DATE)

=> E "ARSENIC"/CN 25

E1 1 ARSENIAN STIBIOLUZONITE/CN

E2 1 ARSENIAN STRONTIAN GAMAGARITE/CN

E3 1 --> ARSENIC/CN

E4 1 ARSENIC (+3 OXIDATION STATE) METHYLTRANSFERASE (ALCANIVORAX

BORKUMENSIS STRAIN SK2)/CN

E5 1 ARSENIC (AS1+)/CN

E6 1 ARSENIC (AS2)/CN

E7 1 ARSENIC (AS3)/CN

E8 1 ARSENIC (AS4)/CN

E9 1 ARSENIC (AS41+)/CN

E10 1 ARSENIC (III)/CN

E11 1 ARSENIC (III) METHYLTRANSFERASE (OIKOPLLEURA DIOICA CLONE

BACOIKO007-10X111)/CN

E12 1 ARSENIC (V) OXIDE/CN

E13 1 ARSENIC 0-0.1, CARBON 0.2, CHROMIUM 0.4-0.6, COPPER 0-0.2, IRON
96-98, MANGANESE 0.6-1, MOLYBDENUM 0.2, NICKEL 0.4-0.8, SILICON 0.2-0.4, VANADIUM
0-0.1/CN

E14 1 ARSENIC 0-0.1, COPPER 99.9-100 (ATOMIC)/CN

E15 1 ARSENIC 0-0.2, CADMIUM 100/CN
E16 1 ARSENIC 0-1, CARBON 7, IRON 79-80, PHOSPHORUS 13 (ATOMIC)/CN
E17 1 ARSENIC 0-1, GERMANIUM 45-55, TELLURIUM 45-55 (ATOMIC)/CN
E18 1 ARSENIC 0-1, NICKEL 99-100 (ATOMIC)/CN
E19 1 ARSENIC 0-1.5, SILVER 98.5-100 (ATOMIC)/CN
E20 1 ARSENIC 0-2, LEAD 98-100/CN
E21 1 ARSENIC 0-2, COPPER 90.5-92.5, INDIUM 7.5 (ATOMIC)/CN
E22 1 ARSENIC 0-21, TIN 79-100 (ATOMIC)/CN
E23 1 ARSENIC 0-22, LEAD 78-100 (ATOMIC)/CN
E24 1 ARSENIC 0-25, BORON 0-25, PALLADIUM 75 (ATOMIC)/CN
E25 1 ARSENIC 0-3.1, SILVER 96.9-100 (ATOMIC)/CN

=> E "CHLOROQUINE"/CN 25

E1 1 CHLOROQUANIL/CN
E2 1 CHLOROQUINE DIPHOSPHATE/CN
E3 1 --> CHLOROQUINE/CN
E4 1 CHLOROQUINE 2,5-DIHYDROXYBENZOATE/CN
E5 1 CHLOROQUINE ARTESUNATE/CN
E6 1 CHLOROQUINE ASCORBATE/CN
E7 1 CHLOROQUINE CHONDROITIN SULFATE/CN
E8 1 CHLOROQUINE DIASCORBATE/CN
E9 1 CHLOROQUINE DIHYDROCHLORIDE/CN
E10 1 CHLOROQUINE DIHYDROGEN PHOSPHATE (1:2) /CN
E11 1 CHLOROQUINE DIROTAKE/CN
E12 1 CHLOROQUINE DIPHOSPHATE/CN
E13 1 CHLOROQUINE DIPHOSPHATE MONOHYDRATE/CN
E14 1 CHLOROQUINE HYDROCHLORIDE/CN
E15 1 CHLOROQUINE MUSTARD/CN
E16 1 CHLOROQUINE N-OXIDE/CN
E17 1 CHLOROQUINE PHOSPHATE/CN
E18 1 CHLOROQUINE PHOSPHATE-PROGUANIL HYDROCHLORIDE MIXT./CN
E19 1 CHLOROQUINE RESISTANCE MARKER PROTEIN (PLASMODIUM FALCIPARUM
STRAIN 3D7 GENE PF14-0463)/CN
E20 1 CHLOROQUINE RESISTANCE PROTEIN (PLASMODIUM FALCIPARUM STRAIN 7G8
GENE CG2)/CN
E21 1 CHLOROQUINE RESISTANCE PROTEIN (PLASMODIUM FALCIPARUM STRAIN HB3
GENE CG2)/CN
E22 1 CHLOROQUINE RESISTANCE TRANSPORTER (CRT)-LIKE PROTEIN
(PLASMODIUM CHABAUDI CLONE AS GENE CG10)/CN
E23 1 CHLOROQUINE RESISTANCE TRANSPORTER (PLASMODIUM FALCIPARUM CLONE
106/1 GENE CRT)/CN
E24 1 CHLOROQUINE RESISTANCE TRANSPORTER (PLASMODIUM FALCIPARUM CLONE
7G8 GENE CRT)/CN
E25 1 CHLOROQUINE RESISTANCE TRANSPORTER (PLASMODIUM FALCIPARUM CLONE
DIV30 GENE CRT)/CN

=> S E3

L2 1 CHLOROQUINE/CN

=> DIS L2 1 IDE

THE ESTIMATED COST FOR THIS REQUEST IS 2.05 U.S. DOLLARS
DO YOU WANT TO CONTINUE WITH THIS REQUEST? (Y)/N:Y

L2 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2009 ACS on STN
RN 54-05-7 REGISTRY

ED Entered STN: 16 Nov 1984

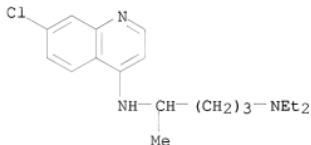
CN 1,4-Pentanediamine, N4-(7-chloro-4-quinolinyl)-N1,N1-diethyl- (CA INDEX
NAME)

OTHER CA INDEX NAMES:

CN Quinoline, 7-chloro-4-[(4-(diethylamino)-1-methylbutyl]amino]- (8CI)

OTHER NAMES:

CN (±)-Chloroquine
 CN 7-Chloro-4-[(4-(diethylamino)-1-methylbutyl]amino]quinoline
 CN Aralen
 CN Atrichin
 CN Bipiquin
 CN Capquin
 CN Chloraqueine
 CN Chlorochin
 CN Chloroquine
 CN NSC 187208
 CN Reumachlor
 CN Ronaqueine
 CN RP 3377
 CN ST 121
 CN ST 121 (pharmaceutical)
 DR 56598-66-4
 MF C18 H26 Cl N3
 CI COM
 LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, BEILSTEIN*, BIOSIS, BIOTECHNO, CA, CABAB, CAPLUS, CASREACT, CBNB, CHEMCATS, CHEMLIST, CIN, CSChem, CSNB, DDFU, DRUGU, EMBASE, HSDB*, IFICDB, IFIPAT, IFIUDB, IMSPRODUCT, IPA, MEDLINE, MRCK*, PIRA, PROMT, PS, RTECS*, SPECINFO, TOXCENTER, USAN, USPAT2, USPATFULL, USPATOLD, VETU
 (*File contains numerically searchable property data)
 Other Sources: EINECS**, WHO
 (**Enter CHEMLIST File for up-to-date regulatory information)



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

5751 REFERENCES IN FILE CA (1907 TO DATE)
94 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
5768 REFERENCES IN FILE CAPLUS (1907 TO DATE)

```
=> index bioscience
FILE 'DRUGMONOG' ACCESS NOT AUTHORIZED
COST IN U.S. DOLLARS
                               SINCE FILE      TOTAL
                               ENTRY SESSION
FULL ESTIMATED COST          16.24    16.46

INDEX 'ADISCTI, ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, ANTE, AQUALINE,
AQUASCI, BIOENG, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, CABA, CAPLUS,
CEABA-VTB, CIN, CONFSCI, CROB, CROPU, DDFB, DDFU, DGENE, DISSABS, DRUGB,
DRUGMONOG2, DRUGL, EMBAL, EMBASE, ...' ENTERED AT 12:14:15 ON 11 NOV 2009
```

68 FILES IN THE FILE LIST IN STNINDEX

Enter SET DETAIL ON to see search term postings or to view

search error messages that display as 0* with SET DETAIL OFF.

=> b reg
COST IN U.S. DOLLARS
SINCE FILE
ENTRY SESSION
0.68 17.14
FULL ESTIMATED COST

FILE 'REGISTRY' ENTERED AT 12:14:19 ON 11 NOV 2009
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STRUCTURE FILE UPDATES: 10 NOV 2009 HIGHEST RN 1192107-32-6
DICTIONARY FILE UPDATES: 10 NOV 2009 HIGHEST RN 1192107-32-6

New CAS Information Use Policies, enter **HELP USAGETERMS** for details.

TSCA INFORMATION NOW CURRENT THROUGH June 26, 2009.

Please note that search-term pricing does apply when conducting SmartSELECT searches.

REGISTRY includes numerically searchable data for experimental and predicted properties as well as tags indicating availability of experimental property data in the original document. For information on property searching in REGISTRY, refer to:

<http://www.cas.org/support/stnqgen/stndoc/properties.html>

=> sel L2 chem
E1 THROUGH E17 ASSIGNED

```
=> index bioscience
FILE 'DRUGMONOG' ACCESS NOT AUTHORIZED
COST IN U.S. DOLLARS          SINCE FILE      TOTAL
                                ENTRY      SESSION
FULL ESTIMATED COST          0.86       18.00

INDEX 'ADISCTI, ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, ANTE, AQUALINE,
AQUASCI, BIOENG, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, CABA, CAPLUS,
CEABA-VTB, CIN, CONFSCI, CROB, CROPU, DDFB, DDFU, DGENE, DISBASS, DRUGB,
DRUGMONOG2, DRUGL, EMBAL, EMBASE' ! ENTERED AT 12:14:33 ON 11 NOV 2009
```

68 FILES IN THE FILE LIST IN STNINDEX

Enter SET DETAIL ON to see search term postings or to view search error messages that display as 0* with SET DETAIL OFF.

```
=> s el-17 or arsen### or retin### (s) lysosom##  
 646  FILE ADISCTI  
 42   FILE ADISINSIGHT  
 533  FILE ADISNEWS  
 4811 FILE AGRICOLA  
 6579 FILE ANABSTR  
 1312 FILE ANTE  
 3701 FILE AQUALINE  
 3461 FILE AQUASCI  
 2140 FILE RIOENG
```

42969 FILE BIOSIS
835 FILE BIOTECHABS
835 FILE BIOTECHDS
4978 FILE BIOTECHNO

13 FILES SEARCHED...
19184 FILE CABA
378054 FILE CAPLUS
1332 FILE CEABA-VTB
1196 FILE CIN
2724 FILE CONFSCI
623 FILE CROPB
771 FILE CROPU
4248 FILE DDFB
8280 FILE DDFU
2533 FILE DGENE

23 FILES SEARCHED...
6276 FILE DISSABS
4248 FILE DRUGB
921 FILE DRUGMONOG2
9040 FILE DRUGU
233 FILE EMBAL
43870 FILE EMBASE
12381 FILE ESBIOBASE
3 FILE FOMAD
2 FILE FOREGE
1241 FILE FROSTI
1954 FILE FSTA

34 FILES SEARCHED...
49738 FILE GENBANK
1585 FILE HEALSAFE
23987 FILE IFIPAT
69 FILE IMSDRUGNEWS
267 FILE IMSPRODUCT
39 FILE IMSRESEARCH
93 FILE KOSMET
11557 FILE LIFESCI
35091 FILE MEDLINE
11652 FILE NTIS
36 FILE NUTRACEUT
610 FILE OCEAN
43096 FILE PASCAL

47 FILES SEARCHED...
18 FILE PCTGEN
42 FILE PHAR
95 FILE PHARMAML
708 FILE PHIN
43225 FILE PROMT
243 FILE PROUSSDR
2 FILE PS
113 FILE RDISCLOSURE
52238 FILE SCISEARCH
6 FILE SYNTHLINE
83726 FILE TOXCENTER
579 FILE USGENE

59 FILES SEARCHED...
111400 FILE USPATFULL
13520 FILE USPATOLD
24709 FILE USPAT2
796 FILE VETB
263 FILE VETU

64 FILES SEARCHED...

5012 FILE WATER
38379 FILE WPIDS
63 FILE WPIFV
38379 FILE WPINDEX

68 FILES HAVE ONE OR MORE ANSWERS, 68 FILES SEARCHED IN STNINDEX

L3 QUE ("(±)-CHLOROQUINE"/BI OR ARALEN/BI OR ARTRICHIN/BI OR BIPIQUIN/BI OR R CAPQUIN/BI OR CHLORAQUINE/BI OR CHLOROCHIN/BI OR CHLOROQUINE/BI OR "NSC 187208"/BI OR REUMACHLOR/BI OR RONAQUINE/BI OR "RP 3377"/BI OR "ST 121 (PHARMACEUTICAL)"/BI OR "ST 121"/BI OR 54-05-7/BI OR 56598-66-4/B I OR "7-CHLORO-4-((4-(DIETHYLAMINO)-1-METHYLBUTYL)AMINO)QUINOLINE"/BI) OR ARSEN### OR RETIN### (S) LYSOSOM##

=> s L3 (s) (disrupt#### or destabiliz### or permea##### or degrad#####)

1 FILE ADISCTI
4 FILE ADISINSIGHT
5 FILE ADISNEWS
150 FILE AGRICOLA
40 FILE ANABSTR
10 FILE ANTE
103 FILE AQUALINE
121 FILE AQUASCI
193 FILE BIOENG
1125 FILE BIOSIS
106 FILE BIOTECHABS
106 FILE BIOTECHDS
664 FILE BIOTECHNO
484 FILE CABA
2676 FILE CAPLUS
46 FILE CEABA-VTB
7 FILE CIN
18 FILE CONFSCI
18 FILES SEARCHED...

8 FILE CROPB
26 FILE CROPU
73 FILE DDFB
263 FILE DDFU
136 FILE DGENE
235 FILE DISSABS
73 FILE DRUGB
415 FILE DRUGU
27 FILES SEARCHED...

7 FILE EMBAL
921 FILE EMBASE
956 FILE ESBIOBASE
17 FILE FROSTI
40 FILE FSTA
983 FILE GENBANK
12 FILE HEALSAFE
182 FILE IFIPAT
1 FILE IMSDRUGNEWS
1 FILE IMSRESEARCH
3 FILE KOSMET
796 FILE LIFESCI
985 FILE MEDLINE
174 FILE NTIS
19 FILE OCEAN
883 FILE PASCAL
47 FILES SEARCHED...

2 FILE PHIN

230 FILE PROMT
5 FILE PROUSDDR
1 FILE RDISCLOSURE
800 FILE SCISEARCH
897 FILE TOXCENTER
2954 FILE USPATFULL
200 FILE USPATOLD
61 FILES SEARCHED...
514 FILE USPAT2
5 FILE VETU
177 FILE WATER
249 FILE WPIDS
1 FILE WPIFV
249 FILE WPINDEX

56 FILES HAVE ONE OR MORE ANSWERS, 68 FILES SEARCHED IN STNINDEX

L4 QUE L3 (S) (DISRUPT##### OR DESTABILIZ### OR PERMEA##### OR DEGRAD#####)

=> s (el-17 or arsen## or retin##) (3a) lysosom#
2 FILE ADISNEWS
17 FILE AGRICOLA
1 FILE ANABSTR
3 FILE AQUASCI
7 FILE BIOENG
506 FILE BIOSIS
8 FILE BIOTECHABS
8 FILE BIOTECHDS
98 FILE BIOTECHNO
40 FILE CABA
607 FILE CAPLUS
5 FILE CONFSCI
19 FILE DDFB
22 FILE DDFU
114 FILE DGENE

23 FILES SEARCHED...
17 FILE DISSABS
19 FILE DRUGB
42 FILE DRUGU
2 FILE EMBAL
348 FILE EMBASE
128 FILE ESBIOBASE
48 FILE GENBANK
10 FILE IFIPAT
1 FILE KOSMET
78 FILE LIFESCI
378 FILE MEDLINE
1 FILE NTIS
91 FILE PASCAL

47 FILES SEARCHED...
1 FILE PHIN
2 FILE PROMT
242 FILE SCISEARCH
271 FILE TOXCENTER
136 FILE USPATFULL
23 FILE USPAT2
1 FILE VETB
4 FILE WPIDS

67 FILES SEARCHED...
4 FILE WPINDEX

```

37 FILES HAVE ONE OR MORE ANSWERS,    68 FILES SEARCHED IN STNINDEX

L5    QUE (E1-17 OR ARSEN### OR RETIN###) (3A) LYSOSOM##

=> s L5 (s) (disrupt### or destabiliz### or permea##### or degrad#####)
    14  FILE BIOSIS
    1  FILE BIOTECHABS
    1  FILE BIOTECHDS
    1  FILE BIOTECHNO
    1  FILE CABA
    19  FILE CAPLUS
    2  FILE DDFU
    22  FILE DGENE
    2  FILE DRUGU
28 FILES SEARCHED...
    13  FILE EMBASE
<-----User Break----->

=> s L5 (5a) (disrupt### or destabiliz### or permea##### or degrad#####)
    12  FILE BIOSIS
    1  FILE BIOTECHABS
    1  FILE BIOTECHDS
    14  FILE CAPLUS
    2  FILE DDFU
    2  FILE DRUGU
    10  FILE EMBASE
    4  FILE ESBIOBASE
    24  FILE GENBANK
37 FILES SEARCHED...
    9  FILE MEDLINE
    5  FILE PASCAL
    9  FILE SCISEARCH
    9  FILE TOXCENTER
    1  FILE USPATFULL
    1  FILE WPIDS
    1  FILE WPINDEX

16 FILES HAVE ONE OR MORE ANSWERS,    68 FILES SEARCHED IN STNINDEX

L6    QUE L5 (5A) (DISRUPT### OR DESTABILIZ### OR PERMEA##### OR DEGRAD#####)

=> d rank
F1          24  GENBANK
F2          14  CAPLUS
F3          12  BIOSIS
F4          10  EMBASE
F5          9   MEDLINE
F6          9   SCISEARCH
F7          9   TOXCENTER
F8          5   PASCAL
F9          4   ESBIOBASE
F10         2   DDFU
F11         2   DRUGU
F12         1   BIOTECHABS
F13         1   BIOTECHDS
F14         1   USPATFULL
F15         1   WPIDS
F16         1   WPINDEX

=> fil f2-f7, f9-f16

```

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	12.24	30.24

FILE 'CAPLUS' ENTERED AT 12:25:25 ON 11 NOV 2009
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FILE 'EMBASE' ENTERED AT 12:25:25 ON 11 NOV 2009
Copyright (c) 2009 Elsevier B.V. All rights reserved.

FILE 'MEDLINE' ENTERED AT 12:25:25 ON 11 NOV 2009

FILE 'SCISEARCH' ENTERED AT 12:25:25 ON 11 NOV 2009
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FILE 'TOXCENTER' ENTERED AT 12:25:25 ON 11 NOV 2009
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FILE 'ESBIOBASE' ENTERED AT 12:25:25 ON 11 NOV 2009
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FILE 'DDFU' ACCESS NOT AUTHORIZED

FILE 'DRUGU' ENTERED AT 12:25:25 ON 11 NOV 2009
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FILE 'BIOTECHABS' ACCESS NOT AUTHORIZED

FILE 'BIOTECHDS' ENTERED AT 12:25:25 ON 11 NOV 2009
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FILE 'USPATFULL' ENTERED AT 12:25:25 ON 11 NOV 2009
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FILE 'WPIDS' ENTERED AT 12:25:25 ON 11 NOV 2009
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FILE 'WPIINDEX' ACCESS NOT AUTHORIZED

=> s L6
L7 72 L6

=> dup rem L7
PROCESSING COMPLETED FOR L7
L8 22 DUP REM L7 (50 DUPLICATES REMOVED)

=> s L8 and py<2005
5 FILES SEARCHED...
L9 16 L8 AND PY<2005

=> d L9 ibib abs 1-16

L9 ANSWER 1 OF 16 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2004:1043363 CAPLUS <<LOGINID::20091111>>
DOCUMENT NUMBER: 142:253721
TITLE: Chloroquine causes lysosomal dysfunction in neural

AUTHOR(S): retina and RPE: Implications for retinopathy
Mahon, G. J.; Anderson, H. R.; Gardiner, T. A.;
McFarlane, S.; Archer, D. B.; Stitt, A. W.
CORPORATE SOURCE: Eye Department, Institute of Clinical Science, Royal
Victoria Hospital, Belfast, UK
SOURCE: Current Eye Research (2004), 28(4), 277-284
CODEN: CEYRDM; ISSN: 0271-3683
PUBLISHER: Taylor & Francis The Netherlands
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Chronic use of chloroquine has been shown to induce numerous pathophysiol. defects in the retina. This drug has the ability to alter pH of intracellular compartments and lysosomal function of the retinal pigment epithelium (RPE) and retinal neurons may constitute the basis of chloroquine retinopathy. The aim of the current study was to investigate pathogenic alterations in retinal cells continuously exposed to chloroquine using appropriate in vivo and in vitro models. Male hooded Lister rats were implanted with osmotic mini pumps which released chloroquine continuously over a period of seven days. The eyes were processed for electron microscopy and ultrastructural abnormalities determined in the neural retina and quantified using stereol. in the retinal pigment epithelium (RPE). RPE were also exposed to chloroquine in vitro and lysosomal pH changes were investigated using a pH sensitive probe. Degradative capacity was also analyzed using FITC labeled rod outer segments (ROS). Chloroquine-treated animals displayed several ultrastructural abnormalities including numerous membranous cytoplasmic bodies (MCBs) in retinal neurons. Cone photoreceptors displayed numerous MCBs although rods did not. The RPE of the treated groups all showed significantly higher nos. of lysosomal associated organelles (LAO) than the control group ($p < 0.001$). The in vitro expts. demonstrated chloroquine-mediated rises in lysosomal pH and an increase in lysosome/phagosome accumulation of ROS in the chloroquine treated group ($p < 0.01$). The current study demonstrates that chloroquine disrupts lysosomal function in retinal neurons and RPE. The evidence presented provides a clear pathogenic basis for the functional defects experienced by patients with chloroquine retinopathy.
OS.CITING REF COUNT: 10 THERE ARE 10 CAPLUS RECORDS THAT CITE THIS RECORD (10 CITINGS)
REFERENCE COUNT: 33 THERE ARE 33 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 2 OF 16 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2003:474355 CAPLUS <>LOGINID::20091111>>
DOCUMENT NUMBER: 139:286273
TITLE: Retinoic acid-induced Golgi apparatus disruption in F2000 fibroblasts: A model for enhanced intracellular retrograde transport
AUTHOR(S): Tzankov, Alexander
CORPORATE SOURCE: Institute of Pathology, University of Innsbruck, Innsbruck, A-6020, Austria
SOURCE: Journal of Biochemistry and Molecular Biology (2003), 36(3), 265-268
PUBLISHER: Biochemical Society of the Republic of Korea
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Retinoic acid (RA) can transform the Golgi apparatus (GA) into a diffuse vacuolar aggregate and increase the toxicity of some immunotoxins that enter into cells by receptor-mediated endocytosis. An ultramorphol. study of the RA-induced GA disruption was performed on F2000 fibroblasts. Cultures were treated with 0.11 to 30 μ M RA for 7-180 min. The

endocytosis of *Limax flavus* agglutinin-peroxidase conjugate (LFA), and the interactions between a phorbol ester (PMA) and RA concerning GA disruption, were examined. Exposure to 0.33 μ M RA for 20 min transformed the GA into vacuolar aggregate. These vacuoles were not involved in endocytosis since they remained unstained after endocytosis of LFA. However, the lysosomes were involved in endocytosis, as they were strongly stained. Therefore, a RA-induced shift towards lysosomal routing of the entered LFA was presumed. Exposure to PMA made cells resistant to the Golgi-disturbing effects of RA, indicating that protein kinase C plays an important role in this process.

OS.CITING REF COUNT: 2 THERE ARE 2 CAPLUS RECORDS THAT CITE THIS RECORD
(2 CITINGS)
REFERENCE COUNT: 28 THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 3 OF 16 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2001:60963 CAPLUS <<LOGINID::20091111>>
DOCUMENT NUMBER: 134:309191
TITLE: Does A2E, a retinoid component of lipofuscin and inhibitor of lysosomal degradative functions, directly affect the activity of lysosomal hydrolases?
AUTHOR(S): Bermann, Marion; Schutt, Florian; Holz, Frank G.; Kopitz, Jürgen
CORPORATE SOURCE: Department of Pathochemistry and General Neurochemistry, Im Neuenheimer Feld 220/221, Germany
SOURCE: Experimental Eye Research (2001), 72(2), 191-195
CODEN: EXERA6; ISSN: 0014-4835
PUBLISHER: Academic Press
DOCUMENT TYPE: Journal
LANGUAGE: English
AB A2E is a retinoid component of lipofuscin and inhibitor of lysosomal degradative functions. It has been suggested that inhibition of the lysosomal degradative function of retinal pigment epithelium (RPE) cells by accumulating A2E is a causative factor in the pathophysiol. of retinal diseases associated with excessive lipofuscin accumulations, including age-related macular degeneration (ARMD) and Stargardt's disease. Therefore, the authors investigated the possibility that A2E was a direct inhibitor of one or more lysosomal hydrolases from cultured RPE cells. The effects of 0.1, 1 and 10 μ M A2E on the activities of these hydrolases were measured in specific enzyme assays. All major classes of lysosomal hydrolases were covered including proteases, lipidases, glycosidases, nucleases, sulfatases, and phosphatases. All enzyme activities were detectable in cultured RPE cells. The results show that A2E, even at 10 μ M, did not cause inhibition of any of the enzymes tested. In conclusion, because all tested enzyme activities remained unaffected by A2E, it is unlikely that a direct inhibition of lysosomal enzymes can explain the pathophysiol. role of A2E in ARMD and related diseases. (c) 2001 Academic Press.

OS.CITING REF COUNT: 16 THERE ARE 16 CAPLUS RECORDS THAT CITE THIS RECORD (16 CITINGS)
REFERENCE COUNT: 24 THERE ARE 24 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 4 OF 16 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 1999:152085 CAPLUS <<LOGINID::20091111>>
DOCUMENT NUMBER: 130:323405
TITLE: Melanin granules of retinal pigment epithelium are connected with the lysosomal degradation pathway
AUTHOR(S): Schraermeyer, Ulrich; Peters, Swaantje; Thumann, Gabriele; Kociok, Norbert; Heimann, Klaus

CORPORATE SOURCE: Department of Vitreoretinal Surgery, University of Cologne, Cologne, 50931, Germany
SOURCE: Experimental Eye Research (1999), 68(2), 237-245
CODEN: EXERA6; ISSN: 0014-4835
PUBLISHER: Academic Press
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Melanosomes are closely related to lysosomes and lipofuscin granules. This paper indicates the potential involvement of lysosomal degradation processes in retinal pigment epithelial (RPE) cells. RPE cells cultured on Bruch's membrane and choroid were fed with indigestible latex beads. The RPE cells from this preparation were treated with chloroquine to investigate whether membrane swelling, typical for lysosomes under this condition, can be induced in melanosomes. To investigate the fate of indigestible material associated with rod outer segments (ROS), gold-labeled ROS were injected transsclerally into the subretinal space of Long Evans rats using a 32 gauge Hamilton syringe. The degradation of labeled ROS was observed after 5 and 12 days by electron microscopy. The following results were observed. Latex particles fuse with the melanin granules of the RPE. Following chloroquine treatment, the membranes of melanin granules fused, and formed large clusters and vacuoles. Gold granules were detected inside both early stage melanosomes and mature melanin granules of the RPE cells 5 and 12 days following subretinal injection of the labeled ROS. Higher nos. of gold granules were predominantly found in immature melanosomes containing still melanofilaments and in small fused mature melanin granules. In conclusion the effect of chloroquine clearly demonstrates that the melanosomes possess active proton pumps which is typical for lysosomes. In RPE cells stressed by overload with rod outer segments or by ingestion of undegradable material (latex beads, gold particles), fusion of these phagosomes with melanosomes of different maturity is more a general rule than an exception. Therefore, melanosomes are connected to lysosomal pathways in RPE cells. (c) 1999 Academic Press.

OS.CITING REF COUNT: 31 THERE ARE 31 CAPLUS RECORDS THAT CITE THIS RECORD (31 CITINGS)
REFERENCE COUNT: 37 THERE ARE 37 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 5 OF 16 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 1995:1005284 CAPLUS <<LOGINID::20091111>>
DOCUMENT NUMBER: 124:44915
ORIGINAL REFERENCE NO.: 124:8211a,8214a
TITLE: Lipidemic effect as a manifestation of chloroquine retinotoxicity
AUTHOR(S): Gaafar, K. M.; Abdel-Khalek, L. R.; El-Sayed, N. K.; Ramadan, G. A.
CORPORATE SOURCE: Dep. Zool., Cairo Univ., Giza, Egypt
SOURCE: Arzneimittel-Forschung (1995), 45(11), 1231-5
CODEN: ARZNAD; ISSN: 0004-4172
PUBLISHER: Cantor
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The effect of long-term treatment of chloroquine (CAS 54-05-7) (20 mg/kg body weight) on serum lipid components and its relation to the retinotoxic effect was studied in albino rats. Chloroquine was found to form lamellar lysosome-like structures within the photoreceptor layer, as well as the pigment epithelium and neuroretinal layers. Biochem., hypolipidemia in the serum was observed mainly due to the decrease in phospholipid portion. It was hypothesized that due to the inhibition of the degradation

process in the defective lysosomes, the retinal cells were denied the re-use of their own phospholipids, and thereby resort to their uptake from the serum.

OS.CITING REF COUNT: 4 THERE ARE 4 CAPLUS RECORDS THAT CITE THIS RECORD
(4 CITINGS)

L9 ANSWER 6 OF 16 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 1995:533430 CAPLUS <<LOGINID::20091111>>
DOCUMENT NUMBER: 123:6438
ORIGINAL REFERENCE NO.: 123:1347a,1350a
TITLE: Enzymic digestion increases permeability of the outer blood-retinal barrier for high-molecular-weight substances
AUTHOR(S): Prunte, Christian; Kain, Hermann L.
CORPORATE SOURCE: University Eye Clinic, Basel, CH-4056, Switz.
SOURCE: Graefe's Archive for Clinical and Experimental Ophthalmology (1995), 233(2), 101-11
CODEN: GACODL; ISSN: 0721-832X
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The purpose of the study was to investigate whether lysosomal enzymes can participate in damaging the outer blood-retinal barrier and to examine the role of glycosaminoglycans in maintaining the barrier function for high-mol.-weight substances. The ciliary artery was cannulated in freshly enucleated pig eyes. Perfusion was performed with buffer (controls), with heparinase (substrate: heparan sulfate), or with lysosomal enzymes freshly prepared from pig retinal pigment epithelium at 36°C, followed by perfusion with the tracer native ferritin (NF) or the marker cationized ferritin (CF). The eyes were examined by electron microscopy. In controls treated with buffer alone, NF was found in high concentration in the lumina of the choroidal capillaries; however, little NF was found in Bruch's membrane (Bsm). The tracer did not penetrate to any extent beyond Bsm. In eyes digested with heparinase or lysosomal enzymes, significantly higher nos. of tracer mols. were found in Bsm. Furthermore, NF penetrated Bsm and was apparent in the subretinal space and also inside retinal pigment epithelial cells, probably due to pinocytosis. The results indicate that heparan sulfate proteoglycan is important for the maintenance of the outer blood-retinal barrier and that lysosomal proteases may participate in damaging this barrier, causing increased permeability to high-mol.-weight substances.

L9 ANSWER 7 OF 16 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 1980:194829 CAPLUS <<LOGINID::20091111>>
DOCUMENT NUMBER: 92:194829
ORIGINAL REFERENCE NO.: 92:31540h,31541a
TITLE: The sources of acid hydrolases for photoreceptor membrane degradation in a grapsid crab
AUTHOR(S): Blest, A. D.; Stowe, Sally; Price, D. G.
CORPORATE SOURCE: Dep. Neurobiol., Aust. Natl. Univ., Canberra, Australia
SOURCE: Cell & Tissue Research (1980), 205(2), 229-44
CODEN: CTSRCS; ISSN: 0302-766X
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Dawn photoreceptor breakdown in *Leptograpsus variegatus* was analyzed at the ultrastructural level. Coated vesicles derived from microvilli were assembled as multivesicular bodies (mvbs), which degraded to multilamellar bodies (mlbs) and were lysed. Cytochem. markers for hydrolases were a F--inhibited β -glycerophosphatase and a F--insensitive p-nitrophenyl phosphatase, with indistinguishable distributions when localized at pH

5.0. These enzymes were injected into the secondary lysosomes from 2 sources. First, immediately after dawn Golgi bodies were highly active, and differentiated a transtubular network, from which tubules and vesicles detached, and could be seen fusing with mvbs and mbs. Saccules derived from the rough endoplasmic reticulum provided a 2nd source and were most often seen in association with late mbs. Both kinds of primary lysosome rarely gave acid phosphatase-pos. responses when free in the cytosol, but did so as they made contact with their secondary lysosomal targets. Lipid droplets and lipofuscin bodies were interpreted as the residual products of breakdown. These results are discussed in relation to previous findings on photoreceptor membrane breakdown in a dinopid spider. Attention is drawn to the implied diversity of organization of lysosomal compartments in receptors which internalize membranes of similar composition

OS.CITING REF COUNT: 2 THERE ARE 2 CAPLUS RECORDS THAT CITE THIS RECORD (2 CITINGS)

L9 ANSWER 8 OF 16 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 1980:175910 CAPLUS <<LOGINID::20091111>>
DOCUMENT NUMBER: 92:175910
ORIGINAL REFERENCE NO.: 92:28415a,28418a
TITLE: Degradation of rhodopsin by a lysosomal fraction of retinal pigment epithelium: Biochemical aspects of the visual process. XLI
AUTHOR(S): Regan, C. M.; De Grip, W. J.; Daemen, F. J. M.; Bonting, S. L.
CORPORATE SOURCE: Dep. Biochem., Univ. Nijmegen, Nijmegen, 6500 HB, Neth.
SOURCE: Experimental Eye Research (1980), 30(2), 183-91
DOCUMENT TYPE: CODEN: EXERA6; ISSN: 0014-4835
LANGUAGE: English
AB The degradation of rhodopsin, present in a photoreceptor membrane preparation, by a

lysosomal fraction of retinal pigment epithelium and by the enzyme cathepsin D was studied. The lysosomal fraction was obtained by subcellular fractionation of bovine pigment epithelium on a linear sucrose gradient. This fractionation procedure showed the presence of 2 populations of lysosomes in the tissue which are thought to represent phagosomes and autophagous lysosomes. Cathepsin D was purified from bovine spleen by affinity chromatog. The lysosomal fraction and cathepsin D degraded nonilluminated and illuminated rod outer segment membranes similarly. However, the proteolytic rates after illumination were somewhat higher than before illumination. During the initial phase of degradation predominantly other membrane proteins than rhodopsin were attacked, whereas the spectral integrity of rhodopsin was retained and no alteration in photolytic behavior was detected. Subsequently, rhodopsin was slowly but completely degraded with concomitant loss of 500 nm absorbance. Na dodecyl sulfate gel electrophoresis showed that in addition to the 37,000-dalton band, which represents intact rhodopsin, new bands of 33,000-34,000 and 25,000-28,000 daltons appeared during the proteolytic reaction. Thus, the retinal pigment epithelium appears both to degrade rhodopsin sequentially to a specific 25,000-28,000-dalton fragment, and to degrade rhodopsin and its fragments nonspecifically to small peptides.

OS.CITING REF COUNT: 13 THERE ARE 13 CAPLUS RECORDS THAT CITE THIS RECORD (13 CITINGS)

L9 ANSWER 9 OF 16 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 1977:14866 CAPLUS <<LOGINID::20091111>>
DOCUMENT NUMBER: 86:14866

ORIGINAL REFERENCE NO.: 86:2423a,2426a
TITLE: Influence of defective circulation of the posterior ciliary arteries on acid hydrolases in the choroid and the retina
AUTHOR(S): Hara, Satoshi; Hayasaka, Seiji; Kikuchi, Tadasu; Mizuno, Katsuyoshi
CORPORATE SOURCE: Sch. Med., Tohoku Univ., Sendai, Japan
SOURCE: Japanese Journal of Ophthalmology (1976), 20(3), 353-60
DOCUMENT TYPE: CODEN: JJOPA7; ISSN: 0021-5155
LANGUAGE: Journal English
AB The influence of ischemia by obstruction of the posterior temporal ciliary arteries on the rabbit retina and choroid was studied using standard biochemical methods. After dissection of the arteries, the unsedimentable activities of acid phosphatase, β -glucuronidase, and cathepsin D from the retina and the choroid increased markedly, while the total activities of these 3 enzymes were either constant or only slightly increased. The retina, whose adjoining choroidal circulation was occluded exptl., showed patchy degeneration. The ischemia followed by occlusive circulation in choroidal vessels perhaps caused the lysosomal particle to disrupt in the retina, and the lysosomal enzymes like acid phosphatase, β -glucuronidase, and cathepsin D were released from the lysosome particle and entered the unsedimentable fraction. Therefore, an autolytic process of the retina and choroid took place. Pathogenesis of some retinal diseases was discussed based on these results.
OS.CITING REF COUNT: 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD
(1 CITINGS)

L9 ANSWER 10 OF 16 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 1970:518275 CAPLUS <<LOGINID::20091111>>
DOCUMENT NUMBER: 73:118275
ORIGINAL REFERENCE NO.: 73:19257a,19260a
TITLE: Changes of lysosomal enzymes during hereditary degeneration and histogenesis of retina in mice. I. Acid phosphatase visualized by azo-dye and lead nitrate methods
AUTHOR(S): Sanyal, Somes
CORPORATE SOURCE: Dep. Anat., Med. Fac. Rotterdam, Rotterdam, Neth.
SOURCE: Histochemie (1970), 23, 207-19
DOCUMENT TYPE: CODEN: HICHAU; ISSN: 0018-2222
LANGUAGE: Journal English
AB In the normal histogenesis of mouse retina, localized distribution of acid phosphatase pos. granules has been seen around the photoreceptor cell nuclei along the outer limiting membrane. These granules disappear during the development of the rod elements. Temporarily increased activity is also seen along the nuclei of the inner layer adjacent to and in the course of the development of the outer and the inner plexiform layers. Within the inner nuclear layer, the cells at the outer and inner rows develop localized acid phosphatase pos. granules which persist in the adult retina. Ganglion cells and the layer of nerve fibers show little change. In the pigment epithelium the enzyme gradually increases. In mice homozygous for the retinal degeneration gene, degenerating photoreceptor cell nuclei, characterized by perinuclear acid phosphatase staining, can be detected before morphol. signs of degeneration. Increased frequency of such nuclei and intensity of staining are recorded with the progress of degeneration. Enzyme activity in the photoreceptor cells, within the inner nuclear layer and in the degenerating photoreceptor cell nuclei, is demonstrable using naphthol substrates but not β -glycerophosphate. Pos. reaction with β -glycerophosphate

is obtained in these sites in the presence of dimethyl sulfoxide. Existence of differential permeability among the retinal lysosomes is tentatively suggested.

L9 ANSWER 11 OF 16 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 1969:104119 CAPLUS <>LOGINID::20091111>>
DOCUMENT NUMBER: 70:104119
ORIGINAL REFERENCE NO.: 70:19427a,19430a
TITLE: Acute effects of high vitamin A (alcohol, aldehyde, and acid) doses on the stability of lysosomes in perfused rat liver
AUTHOR(S): Frimmer, Max; Gries, J.; Waldvogel, G.
CORPORATE SOURCE: Inst. Pharmakol. Toxikol., Justus-Liebig-Univ. Giessen, Giessen, Fed. Rep. Ger.
SOURCE: Internationale Zeitschrift fuer Vitaminforschung (1968), 38(5), 454-8
DOCUMENT TYPE: CODEN: IZVIAK; ISSN: 0020-9406
LANGUAGE: Journal German
AB Addition of 2500, 5000, 7500, or 10,000 immunization units of vitamin A acid (retinoic acid), vitamin A alc. (retinol), or vitamin A aldehyde (retinal) to the rat liver perfusate induced the release of β -glucuronidase from the liver cells. At the high concns. of the vitamin in the whole liver, a toxic effect was exerted on the lysosomal and other cellular membranes. At the lower doses, retinoic acid, retinol, and retinal induced the release of approx. equal quantities of β -glucuronidase.

L9 ANSWER 12 OF 16 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
ACCESSION NUMBER: 2004:226033 BIOSIS <>LOGINID::20091111>>
DOCUMENT NUMBER: PREV200400221138
TITLE: Effects of lysosomal A2E on lipid degradation in RPE.
AUTHOR(S): Rodriguez-Boulan, Enrique [Reprint Author]; Lakkaraju, Aparna; Leung, Larry; Finemann, Silvia
CORPORATE SOURCE: Weill Medical College, Cornell University, New York, NY, USA
SOURCE: adc2001@mail.med.cornell.edu Graefe's Archive for Clinical and Experimental Ophthalmology, (January 2004) Vol. 242, No. 1, pp. 60. print.
Meeting Info.: First Workshop on Cell Transplantation in Age-Related Macular Degeneration. Cologne, Germany. September 11-14, 2003.
DOCUMENT TYPE: CODEN: GACODL. ISSN: 0721-832X.
CONFERENCE: Conference; (Meeting)
CONFERENCE ABSTRACT: Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 21 Apr 2004
Last Updated on STN: 21 Apr 2004

L9 ANSWER 13 OF 16 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
ACCESSION NUMBER: 1999:188045 BIOSIS <>LOGINID::20091111>>
DOCUMENT NUMBER: PREV199900188045
TITLE: Inhibition of lysosomal degradative functions in RPE cells by a retinoid component of lipofuscin.
AUTHOR(S): Holz, Frank G. [Reprint author]; Schuett, Florian; Kopitz, Juergen; Eldred, Graig E.; Kruse, Friedrich E.; Voelcker, Hans E.; Cantz, Michael
CORPORATE SOURCE: Department of Ophthalmology, University of Heidelberg, Im Neuenheimer Feld 400, D-69120, Heidelberg, Germany

SOURCE: IOVS, (March, 1999) Vol. 40, No. 3, pp. 737-743.
print.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 5 May 1999
Last Updated on STN: 5 May 1999
AB Purpose. To investigate the effect of the lipofuscin component N-retinylidene-N-retinylethanolamine (A2-E) on degradative functions of lysosomes in human retinal pigment epithelial (RPE) cells and to evaluate its mechanism of action. Methods. A2-E was coupled to low-density lipoprotein (LDL). Human RPE cell cultures were loaded with the A2-E/LDL complex, and controls were run with medium containing LDL alone. To determine whether A2-E accumulated in lysosomes, cells were fractionated in a Percoll gradient, and protein degradation was determined by metabolic labeling and measurement of the release of low-molecular-weight radioactivity. Lysosomal degradation was distinguished from nonlysosomal degradation by inclusion of NH4Cl in the medium. The metabolism of sulfated glycosaminoglycans was studied by radiosulfate incorporation in pulse-chase experiments. Intralysosomal pH was determined using a fluorescent lysosomotropic pH indicator. Results. A2-E accumulated almost exclusively in the lysosomal compartment. Lysosomal protein degradation was reduced in a dose-dependent fashion in A2-E-treated cells. The selectivity of A2-E on lysosomal function was demonstrated by its lack of effect on degradation of extralyosomal protein. Lysosomal glycosaminoglycan catabolism of RPE cells was also strongly inhibited by A2-E. Lysosomal pH was increased by A2-E. Conclusions. The findings indicate that accumulation of A2-E in RPE cells interferes with lysosomal functions as exemplified by its inhibitory effect on protein and glycosaminoglycan catabolic pathways. The quaternary amine character of the A2-E apparently causes a perturbation of the acidic intralysosomal milieu, resulting in diminished hydrolase action and consequent accumulation of undegraded material. Such mechanism could be operative in retinal diseases associated with excessive lipofuscin accumulation including age-related macular degeneration.

L9 ANSWER 14 OF 16 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
ACCESSION NUMBER: 1998:242335 BIOSIS <>LOGINID::20091111>>
DOCUMENT NUMBER: PREV199800242335
TITLE: A lipofuscin compound (A2-E) inhibits lysosomal degradative function in human RPE-cells.
AUTHOR(S): Schutt, F. [Reprint author]; Holz, F. G. [Reprint author]; Kopitz, J.; Kruse, F. E. [Reprint author]; Voelcker, H. E. [Reprint author]
CORPORATE SOURCE: Univ. Heidelberg, Dep. Ophthalmol., Heidelberg, Germany
SOURCE: IOVS, (March 15, 1998) Vol. 39, No. 4, pp. S729.
print.
Meeting Info.: Annual Meeting of the Association for Research in Vision and Ophthalmology. Fort Lauderdale, Florida, USA. May 10-15, 1998. Association for Research in Vision and Ophthalmology.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
Conference; (Meeting Poster)
LANGUAGE: English
ENTRY DATE: Entered STN: 4 Jun 1998
Last Updated on STN: 4 Jun 1998

L9 ANSWER 15 OF 16 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
ACCESSION NUMBER: 1971:42172 BIOSIS <>LOGINID::20091111>>

DOCUMENT NUMBER: PREV197107042172; BR07:42172
TITLE: AN INVESTIGATION INTO THE STRUCTURAL INTEGRITY OF LYSOSOMES
AND ITS EFFECT IN THE NORMAL AND DYSTROPHIC RAT RETINA.
AUTHOR(S): BURDEN E M; READING H W; YATES C M
SOURCE: Experimental Eye Research, (1971) Vol. 11, No. 1,
pp. 140.
CODEN: EXERA6. ISSN: 0014-4835.
DOCUMENT TYPE: Article
FILE SEGMENT: BR
LANGUAGE: Unavailable

L9 ANSWER 16 OF 16 Elsevier Biobase COPYRIGHT 2009 Elsevier Science B.V. on
STN

ACCESSION NUMBER: 1999056348 ESBIOBASE <>LOGINID::20091111>>
TITLE: Inhibition of lysosomal degradative functions in RPE
cells by a retinoid component of lipofuscin
AUTHOR(S): Holz, Frank G.; Schutt, Florian; Kruse, Friedrich E.;
Volcker, Hans E.; Kopitz, Jürgen; Cantz, Michael;
Eldred, Graig E.
CORPORATE SOURCE: Holz, Frank G.; Schutt, Florian; Kruse, Friedrich E.;
Volcker, Hans E. (Department of Ophthalmology,
University of Heidelberg, Im Neuenheimer Feld 400,
Heidelberg (DE)); Holz, Frank G. (Department of
Ophthalmology, University of Heidelberg, Im
Neuenheimer Feld 400, D-69120 Heidelberg (DE)); Kopitz,
Jürgen; Cantz, Michael (Inst. of
Pathochemistry/Neurochem., University of Heidelberg,
Heidelberg (DE)); Eldred, Graig E.
SOURCE: Investigative Ophthalmology and Visual Science
(1999) Volume 40, Number 3, pp. 737-743, 41
refs.
CODEN: IOVSDA ISSN: 0146-0404

COUNTRY OF PUBLICATION: United States of America
DOCUMENT TYPE: Journal; Article
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 31 Jan 2009
Last updated on STN: 31 Jan 2009

AN 1999056348 ESBIOBASE <>LOGINID::20091111>>
AB PURPOSE. To investigate the effect of the lipofuscin component N-
retinylidene-N-retinylethanolamine (A2-E) on degradative
functions of lysosomes in human retinal pigment
epithelial (RPE) cells and to evaluate its mechanism of action. METHODS.
A2-E was coupled to low-density lipoprotein (LDL). Human RPE cell
cultures were loaded with the A2-E/LDL complex, and controls were run
with medium containing LDL alone. To determine whether A2-E accumulated
in lysosomes, cells were fractionated in a Percoll gradient, and protein
degradation was determined by metabolic labeling and measurement of the
release of low-molecular-weight radioactivity. Lysosomal degradation was
distinguished from nonlysosomal degradation by inclusion of NH₄Cl in
the medium. The metabolism of sulfated glycosaminoglycans was studied by
radiosulfate incorporation in pulse-chase experiments. Intralysosomal pH
was determined using a fluorescent lysosomotropic pH indicator. RESULTS.
A2-E accumulated almost exclusively in the lysosomal compartment.
Lysosomal protein degradation was reduced in a dose-dependent fashion in
A2-E-treated cells. The selectivity of A2-E on lysosomal function was
demonstrated by its lack of effect on degradation of extralysosomal
protein. Lysosomal glycosaminoglycan catabolism of RPE cells was also
strongly inhibited by A2-E. Lysosomal pH was increased by A2-E.
CONCLUSIONS. The findings indicate that accumulation of A2-E in RPE
cells interferes with lysosomal functions as exemplified by its

inhibitory effect on protein and glycosaminoglycan catabolic pathways. The quaternary amine character of the A2-E apparently causes a perturbation of the acidic intralysosomal milieu, resulting in diminished hydrolase action and consequent accumulation of undegraded material. Such mechanism could be operative in retinal diseases associated with excessive lipofuscin accumulation including age-related macular degeneration.

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